DISSERTATION
ON

SURGICAL OUTCOME ANALYSIS IN SALIVARY GLAND SWELLINGS

M.S. DEGREE EXAMINATION
BRANCH I
(GENERAL SURGERY)

THANJAVUR MEDICAL COLLEGE

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CERTIFICATE

This is to certify that this dissertation entitled ***"Surgical outcome analysis in salivary gland swellings"*** is the bonafide record of original work done by **Dr.P.Balaraman** in partial fulfillment of the university requirements for M.S. General surgery(Branch I) examination to be held in March 2008

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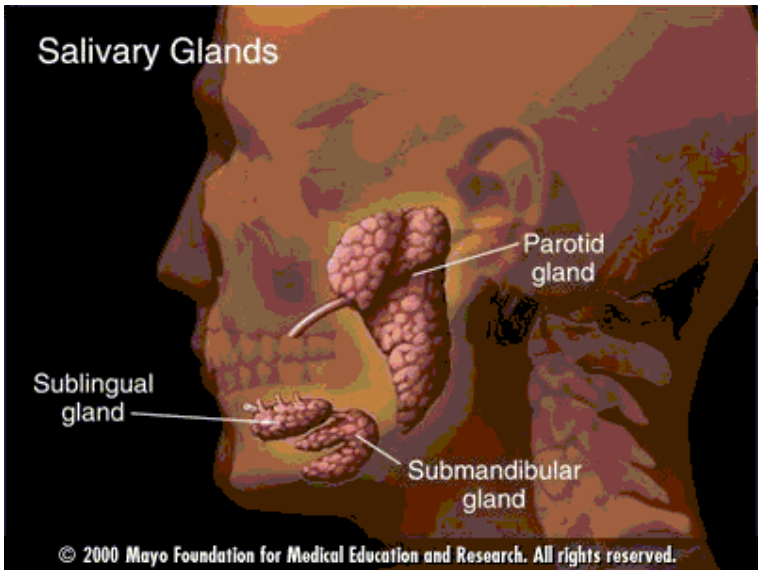
INTRODUCTION



Salivary gland lesions are among an interesting array of pathologies to the surgeon ranging from congenital lesions to neoplasms. These lesions comprise only about 1.5–3% of the surgical admissions but do have an interesting role for the surgeon to play to ensure the recovery of these patients off their symptoms. This craftful field of surgery is explored in this study and the relevant medical literature is being presented here.

History of the Procedures: The anatomy of the parotid gland and the role of the main ducts were described in the mid-17th century. The earliest references to "para-auricular swellings," as the Greeks called them, described the findings associated with calculi and inflammation.

From 1650-1750, salivary gland surgery was limited to the treatment of ranulas and oral calculi. The concept of surgical excision of a parotid tumor has been attributed to **Bertrandi** in 1802. The initial applications of this surgery included an extensive approach, causing serious disfiguration and disability.

By approximately 1850, the focus shifted toward dissection and the intimate relationship between the facial nerve and the parotid gland. Attempts were made to perform the surgery with nerve preservation. **John C. Warren, MD**, was the first to use ether inhalation anesthesia during his resection of a parotid tumor in Boston in 1846. In 1892, **Codreanu** (a Romanian native) performed the first total parotidectomy with facial nerve preservation. Grafting of the facial nerve after resection was attempted in the early 1950s.

Beahrs and Adson (1958) eloquently described the relevant anatomy and surgical technique of current parotid gland surgery. They stressed surgical landmarks for avoiding injury to the facial nerve and advocated complete removal of the superficial portion of the parotid gland for noninvasive lesions confined to that portion of the gland.

Review of literature

The salivary gland system consists of a network of major and minor secretory glands and ducts, the function of which is to keep the oral cavity moist and lubricated and dissolve water soluble compounds as well as begin the digestion of starches.

The salivary system consists of three pairs of major salivary glands- parotid, submandibular and sublingual and hundreds of minor salivary glands present beneath the mucosa of the upper aerodigestive tract starting from the nasal cavity, paranasal sinuses, nasopharynx, oral cavity, oropharynx, hypopharynx and larynx. Embryologically, the salivary glands are tubuloacinar glands arising from the ectodermal and endodermal invaginations. Histologically, major and minor salivary glands consist of two basic cell types- serous and mucous. The serous cells are polyhedral and form globular acini located at the end of microscopic ducts. Serous secretions from these acini empty into smaller duct systems which lead to branching larger duct systems and eventually into the main excretory duct. Mucous cells are cuboidal cells and also form acini. Approximately 1500ml of mucoserous fluid is produced per day, of which 90% is from parotid and submandibular, 5% from sublingual and remainder from minor salivary glands.

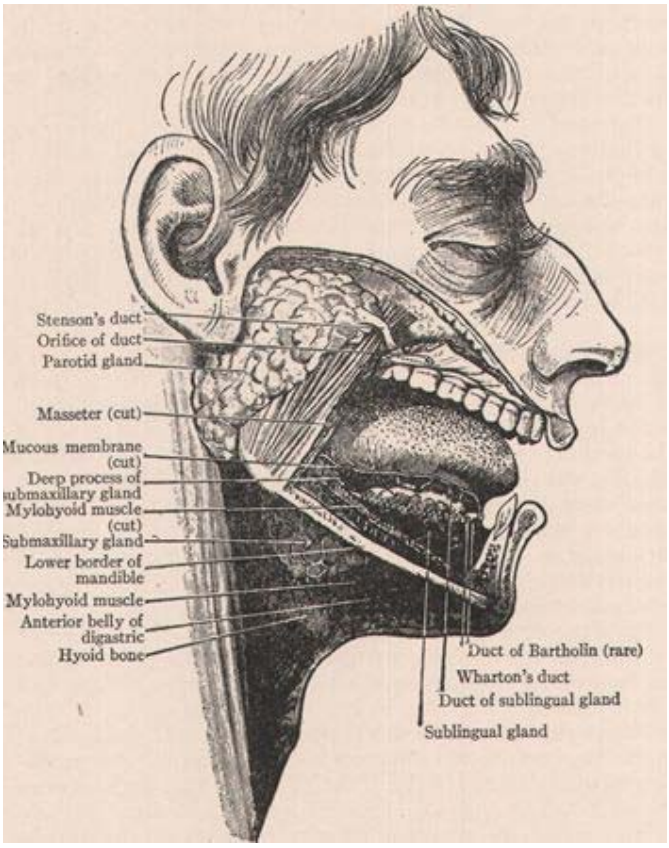
The pathologies affecting these glands can be broadly classified into neoplasms and non-neoplastic lesions which are further sub classified into inflammatory, autoimmune, infectious, traumatic , congenital and other rare lesions. The role of surgical cure in salivary glands is well known and proven beyond doubt in cases of neoplasms and inflammatory swellings, which forms the principal concern of this study.

Surgical Anatomy

Parotid gland:

The parotid gland is the largest of the salivary glands which lies between the zygomatic arch and the angle of the mandible. the parotid compartment is a space that contains the parotid gland, facial nerve and its branches, sensory and autonomic nerves, external carotid artery and its branches, posterior facial vein and lymphatics. About 80% of the gland lies on the outer surface of the masseter muscle and the ramus and the angle of the mandible and the remaining 20% of the gland extends medially through the stylomandibular tunnel formed ventrally by the posterior edge of the mandibular ramus, dorsally by the anterior borders of sternocleidomastoid muscle and the posterior belly of the digastric muscle and more deeply and dorsally by the stylomandibular ligament, and this ligament separates parotid from the submandibular gland. The retro mandibular portion of the parotid lies in the prestyloid compartment of the para-pharyngeal space, being anterior to the styloid process and its musculature, the carotid sheath and the cranial nerves IX, X, XI,XII. Hence a parotid tumour may appear intra orally pushing the soft palate and tonsillar fossa anteromedially. The constricted area of the gland between the ramus of the mandible and posterior belly of digastric connecting the superficial and deep portions is called the “isthmus”. The average dimension of the gland in the cranio-caudal direction is 5.8cm and in

the dorso-ventral direction is 3.4 cm. The gland tends to be slightly smaller in women.



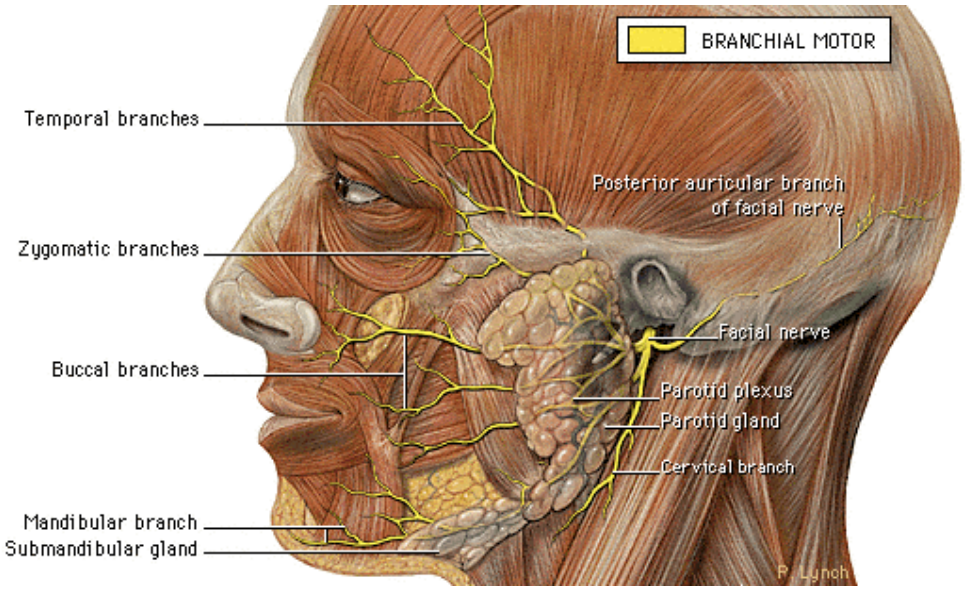
Although not found in all glands, five processes of the parotid gland have been described. Three of them are superficial, which are the condylar, meatal and posterior processes. And the deep processes are glenoid and stylomandibular. Because of the multiple processes and the limitations imposed by an intact facial nerve, it is nearly impossible to remove all parotid tissue even when a total parotidectomy is attempted. The parotid duct emanates from the anterior aspect of the superficial component of the gland, courses anteriorly on the lateral surface of the masseter muscle and buccal fat pad, and then turns medially almost at right angle, piercing the buccinator at the level of the second maxillary molar tooth. The general course of the parotid duct is in the

direction paralleling the zygomatic arch, traversing approximately 1 cm inferior to the arch. Stensen's duct passes over the masseter, may receive the duct of an accessory parotid gland, found in about 20% overlying the masseter muscle, usually cranial to the Stensen's duct.

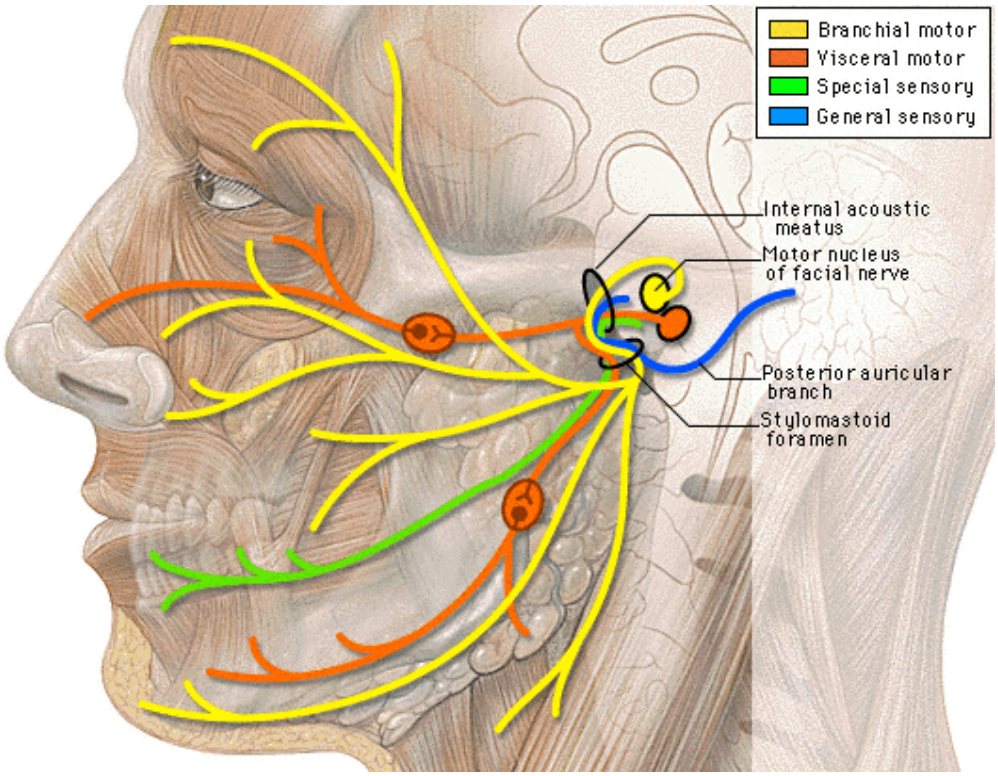
NEUROVASCULAR ANATOMY:

The greater auricular, auriculo temporal and facial nerves traverse the most superficial portion of the parotid gland(mylohyoid neural compartment). From the inception of the surgery, usually the first neural structure encountered is the greater auricular nerve when the skin flaps are raised. This nerve divides into anterior and posterior branches. Preservation of the posterior division of the nerve is possible during removal of smaller parotid tumours and those located away from the tail of the parotid gland.

The auriculo temporal nerve , a branch of the V cranial nerve, is found in the posterior aspect of the gland, coursing superiorly with the superficial temporal artery and vein. It is a sensory nerve to the temporal region and carries parasympathetic fibres to the parotid gland from the otic ganglion.



The facial nerve exits the stylomastoid foramen from its intra temporal course. After the nerve exits the stylomastoid foramen, it gives off immediate branches to the stylohyoid, posterior belly of digastric, and postauricular muscles before its transition to the pes anserinus. The facial nerve enters the parotid gland on its posterior surface prior to the formation of the pes anserinus, about 1.3 cm from the stylomastoid formaen. From the pes, two major portions of the facial nerve are formed-the cervicofacial and temporofacial divisions. The cervico facial division gives rise to marginal mandibular and



cervical branches, and the temporofacial division gives rise to the temporal, zygomatic and buccal branches. There are six types of branching patterns being described for the facial nerve, none of them being the predominant one. The surgeon should keep in mind that the there is a tendency for cross-innervation between the buccal and zygomatic branches. During parotidectomy, each branch of the nerve is traced and the parotid tissue lateral to the branches(in superficial parotidectomy) and both lateral and medial to the branches(in total parotidectomy) is removed.

The veins traverse the middle portion of the gland deep to facial nerve. The superficial temporal vein unites with the internal maxillary vein to form the posterior facial vein, which branches into anterior and posterior divisions. The anterior branch joins the anterior facial vein to form the common facial vein,

whereas the posterior division unites with the posterior auricular vein to form the external jugular vein. On its descent, the external jugular vein is found on the lateral aspect of the sternocleidomastoid muscle , anterior to the greater auricular nerve. This anatomic arrangement is often used to help locate the proximal portion of the nerve for grafting purposes.

The external carotid artery and its terminal branches, the internal maxillary and the superficial temporal arteries, traverse the deep portion of the parotid gland(arterial compartment). The superficial temporal artery ascends between the mandibular condyle and external auditory canal. Precise localization of this artery occurs just at the root of the zygoma anterior to the auricle. The internal maxillary artery, which may be encountered during total parotidectomy or removal of deep lobe tumours in the para-pharyngeal space, is found between the mandibular ramus and sphenomandibular ligament, coursing in close proximity to the lateral pterygoid muscle and heading toward the pterygopalatine fossa.

Superficial temporal vessels provide blood supply to the temporo-parietal fascial flap, which can be used for recontouring defects in the parotid bed. Internal maxillary vessels and the deep temporal branches should be preserved whenever possible, as they provide blood supply to the temporalis muscle which is often used for dynamic rehabilitation of facial paralysis.

The parotid gland is associated with a rich network of lymphatics. During embryogenesis, the parotid anlagen is first to develop but it becomes encapsulated after the submandibular and sublingual glands. The lymphatic system develops from the mesoderm prior to parotid gland encapsulation, and after the submandibular and sublingual glands encapsulate. This delayed encapsulation of the parotid gland is critical because it results in the entrapment of the lymph nodes and lymphatic channels within the glandular parenchyma. In addition, during encapsulation of the intraparotid and periparotid lymphnodes, salivary epithelial cells can be included within these nodes. It is this special embryogenesis of the parotid gland that is believed to play a roe in the development of warthin's tumours, and possibly lymphoepithelial cysts. The pre auricular and infra-auricular lymph nodes constitute the para-parotid group, which drain the temporal region, of the scalp and auricle. The intraparotid nodes receive lymphatics from the posterior nasopharynx, soft palate and ear. Hyperplastic changes or neoplastic involvement of these nodes may manifest as parotid mass.

Parotidomassteric fascia

The superficial or anterior layer of the deep cervical fascia extends from its attachment on the hyoid bone upward to the mandible, and from the anterior border of the sternocleidomastoid upward to the mandible and zygomatic arch.

More posteriorly, at the level of the submandibular gland, the superficial layer splits to form a capsule about this gland, still more posteriorly about the insertions of the masseter and medial pterygoid muscles, it splits to enclose these muscles and the intervening angle and ramus of the mandible, one portion of the masseteric fascia, following the external surface of the masseter to the zygomatic arch and the other portion following the internal surface of the medial pterygoid to the pterygoid plate.

Finally, behind the angle of the jaw and anterior to the sternocleidomastoid, this layer of fascia passes upward towards the zygomatic arch as the parotid fascia. Grodinsky and Holyoke described it as splitting to form a capsule about the parotid gland before attaching to the zygomatic arch. The potential spaces formed by the splitting of the superficial layer of fascia are all closed spaces.

As in the case of the submandibular gland, the parotid gland is attached to its surrounding fascia, and the parotid “space” is therefore not so much anatomical as a clinical one. The density, lack of elasticity and the compartmentalization of the fascia prevent stretching of the capsule to accommodate a suppurative or other rapidly expanding intraparotid process. The deep portion of the parotid fascia that contributes to the stylomandibular ligament and passes downward to fuse with the fascia of the posterior belly of the digastric

and the angle of the mandible serves to separate the parotid gland from the submandibular gland.

Lymphatic drainage

Parotid gland contains two groups of lymphnodes:

1.Superficial group: they are situated under the superficial parotid fascia. The pre-auricular node belongs to this group. They drain lacrimal gland, eyelid, cheeks, root of nose, external auditory canal, scalp and pinna.

2.Deep group: they are situated in the substance of the gland. Apart from parotid gland, lymphatics from external auditory canal, middle ear, soft palate and post-nasal cavities drain into these groups of nodes.

Both superficial and deep groups drain into the upper deep cervical nodes.

The submandibular gland

The submandibular gland possesses a superficial and deep component(lobes), which wrap around the posterior border of mylohyoid muscle. It

is ensheathed by the superficial layer of the deep cervical fascia, like the parotid gland. The main excretory duct of the gland(Wharton's duct) emanates from the deep component of the gland and courses between the hyoglossus/genioglossus muscles and the inner surface of the mandible, coursing toward the floor of the mouth, it ends as a papilla adjacent to the lingual frenulum in the floor of the mouth. The duct at first lies medial to the lingual nerve, but then as the lingual nerve loops down, the duct assumes a position lateral to the lingual nerve. The distal hypoglossal nerve is situated between the submandibular gland and the hyoglossus muscle above the hyoid bone. It is located deep to the posterior belly of the digastric muscle and stylohyoid muscle prior to entering the submandibular triangle. Identification of the submandibular duct, lingual nerve, and hypoglossal nerves is mandatory prior to the removal of the submandibular gland.

The marginal mandibular branch of the VII nerve courses directly over the submandibular gland in the plane over the gland and beneath the platysma muscle layer in a significant percentage of people. It may be encountered during transcervical approach to the gland and should be identified meticulously and preserved. Injury to this single nerve will cause a large and noticeable degree of facial asymmetry.

Facial artery courses deep to the posterior belly of digastric and stylohyoid muscles and under the submandibular gland, then traverses between the

submandibular gland and mandible to ascend toward the face. The facial vein courses in the general direction of the facial artery , but the major distinction is that the facial vein runs on the lateral surface of the gland. The facial vein also runs under the marginal mandibular nerve. In fact one maneuver to preserve the marginal mandibular nerve is to ligate the facial vein 2 to 3 cm below the mandible and lift the tissues above the ligated vein and then the nerve will be lifted superiorly with the vein.

Lymphatic drainage is to the submandibular nodes and then to the jugular chain.

The sublingual and minor salivary glands

The sublingual gland is located undercover of the mandible. It is covered by the thin mucosa of the floor of the mouth, resting on the genioglossus muscle. The gland possesses 8 to 20 smaller ducts (duct of Rivinus) which empty into the floor of the mouth. Occasionally, the anteriorly located ducts may join to empty into a larger duct (Bartholin's duct) adjacent or into the sublingual caruncle along with the submandibular duct.

The minor salivary glands number in the range of 600 to 1000 and spread throughout the upper aerodigestive tract. Areas of concentration include the posterior palatal, buccal, labial and lingual regions.

The para-pharyngeal space

Understanding the anatomy of the Para pharyngeal space is crucial for removal of deep lobe parotid tumours. If one could imagine the Para pharyngeal space taking on the shape of an inverted pyramid, the base of the pyramid will be resting along the skull base and the apex directed inferiorly at the greater cornu of the hyoid bone. The medial border of the Para pharyngeal space is the muscular pharyngeal wall, and the lateral border is the medial pterygoid muscle and mandibular ramus. The normal contents of the space include fat, the internal maxillary artery, ascending pharyngeal artery and pharyngeal venous plexus. Deep lobe parotid tumours will extend into the space by growing between the stylomandibular ligament and bony mandible, taking the pathway of least resistance.

The Para pharyngeal space is divided into pre and post styloid compartments by an imaginary line, which is appreciated best on CT scan, from the styloid process to the medial pterygoid plate. Pre operative determination of the growth characteristics and extension of the tumour within the Para pharyngeal space by axial imaging may provide pre operative clues to the pathology of the tumour.

Histiogenesis of salivary gland neoplasms

The mature salivary gland unit is made up of serous or mucous acini that lead to an intercalated duct, which in turn is connected to a striated duct that finally empties into an extra lobular excretory duct. Myo epithelial cells are located around the periphery of the acini and the intercalated duct.

Theories of tumour histiogenesis:

1.Multi cellular theory:

The genesis of neoplasms from their adult differentiated counterparts of the salivary gland unit e.g.. acinic cell carcinomas would originate from acinar cells, oncocytic tumours from striated duct cells, squamous cell carcinomas and mucoepidermoid carcinomas from excretory duct cells, and all other adenomas and adenocarcinomas from intercalated duct cells.

2.Bi cellular theory:

Neoplasms generate from the two undifferentiated reserve cells called excretory duct reserve cell and the intercalated duct reserve cell. This theory is more plausible because it does not require dedifferentiation of already highly specialized cells such as acinar and striated duct cells.

Some of the factors which are considered as risk factors for the development of salivary gland neoplasms are 1. low dose radiation – both benign and malignant neoplasms

2. smoking – associated with the development of warthin's tumour

3. chromosomal anomalies – allelic loss on chromosome 12q in pleomorphic adenoma, translocation breakpoint at 12q & 15 in pleomorphic adenoma, 8q & 12 translocation involving PLAG 1 (a zinc finger protein)

4. exposure to silica dust and parotid cancer

Differential diagnoses of salivary gland swellings include:

1. **<u>Non-neoplastic disorders</u>**

   Infectious

   - Acute sialadenitis
   - Chronic recurrent sialadenitis
   - Viral parotitis
     i. Paramyxovirus
     ii. CMV
     iii. Coxsackie virus
   - Granulomatous parotitis
     i. Tuberculosis
     ii. Cat scratch disease
   - Actinomycosis

   Non-infectious

   - Sialectasis
   - Sialolithiasis
   - Sjogren's syndrome
   - Sarcoidosis(uveoparotid fever of Heerfordt)
   - Benign lymphoepithelial lesion

- Radiation sialadenitis
- Ptyalism
- Sialadenosis
    i. Vitamin deficiency
    ii. Malnutrition
    iii. Bulimia
    iv. Diabetes
    v. Hypothyroidism
    vi. Alcoholic cirrhosis
    vii. Drugs – thiourea, clonidine, alpha-methyl dopa
    viii. Hemangioma
    ix. Cysts
    x. Chronic sclerosing sialadenitis of submandibular gland(Kuttner tumour)
    xi. Necrotizing sialometaplasia

2. <u>Neoplastic disorders</u>

Benign

- Pleomorphic adenoma
- Monomorphic adenoma
    i. Basal cell adenoma
    ii. Glycogen – rich adenoma
    iii. Clear cell adenoma

iv. others

- Warthin’s tumour
- Oncocytoma
- Sebaceous adenoma
- Sebaceous lymphadenoma
- Papillary ductal adenoma
- Hemangioma

Malignant

- Mucoepidermoid carcinoma
- Acinic cell carcinoma
- Adenocarcinoma
- Adenoid cystic carcinoma
- Malignant mixed tumour
- Squamous cell carcinoma
- Malignant oncocytoma
- Lymphoma
- Metastases
    i. Melanoma
    ii. Squamous cell carcinoma
    iii. Metastases to intra parotid lymphnodes

History, physical examination and investigations

Patients with salivary gland disorders usually present with asymptomatic, slowly enlarging masses. Pain related to a salivary gland neoplasm does not confer a higher risk of malignancy and pain is a consistent feature of inflammatory lesions, related to meals in cases of obstruction to the salivary flow especially in sialolithiasis. Associated infection, hemorrhage in cases of tumours may produce pain. The neoplastic lesions are commoner in the tail of the parotid and submandibular gland neoplasms present as enlargement of the gland itself, whereas minor salivary gland neoplasms present as submucosal painless masses on the palate or in the floor of the mouth. They may rarely masquerade as paranasal sinus neoplasms causing obstructive symptoms of sinusitis, nasal obstruction,epistaxis and others. Para pharyngeal space neoplasms are often diagnosed late in their evolution because of the lack of presenting symptoms until a large size has been attained. Dysphagia or muffled ‘hot potato’ voice may be the first indication of the presence of tumour in the Para pharyngeal space. A thorough general medical history is elicited to provide clues to systemic inflammatory or infectious etiologies of salivary gland enlargement that may be mistaken for a neoplastic process.

Physical examination of salivary gland masses assumes that a thorough general head and neck examination is performed. Facial nerve function is noted carefully and documented. Rapid growth, fixation to adjacent structures, associated

cervical adenopathy, ulceration and facial or other nerve involvement suggest underlying malignancy.

Fine needle aspiration cytology is a simple and reliable method for obtaining diagnosis of a salivary gland neoplasm. Eneroth et al first reported FNAC of a salivary gland tumour..(1967). The sensitivity and specificity of FNAC for salivary gland tumours reported in literature range from a high of 99% sensitivity and 100% specificity (Bhatia et al..) to a low of 90%sensitivity and 75% specificity(Cohen et al..) in general, however, recent series document a trend toward a higher degree of sensitivity and specificity. A higher degree of accuracy has been reported for benign tumours. Mucoepidermoid carcinomas appear to be the most difficult to diagnose by FNAC. To improve the diagnostic accuracy of FNAC, special immunohistochemical stains such as glial fibrillary acidic protein for pleomorphic adenomas and silver staining of nucleolar organizer regions have been used. this investigation is essentially free of complications, though there are reports of hemorrhage and necrosis of the tumour following aspiration in case of Warthin's tumour and lymphoma, the occurrence of which is extremely rare. And many specific reports have alleviated the concerns over needle track seeding.

CT scan is an excellent imaging modality for salivary gland lesions and is of value for a guided FNAC in case of deep lobe lesions or relatively inaccessible lesions. A CT scan can better define the extent of nodal metastases and infiltration into vital neurovascular structures in malignant lesions, and CT is more valuable

than MRI in case of inflammatory disorders. MRI , Tc99m scan, Gallium scan, USG and sialography are other modalities which may be of immense help in certain difficult cases but not routinely mandatory given the cost-benefit ratio. And the role of open biopsy in cases of salivary gland lesions is controversial and very minimal, as for example in cases of doubtful malignancy and a lesion which is discordant between clinical and cytological features.

Clinical features:

The various disorders that affect salivary glands and their clinical and pathological features are beyond the scope of this literature review and only salient features of relevant pathological diagnoses are to be illustrated in brief.

Non-neoplastic lesions:

Sialadenosis: The salivary gland can enlarge for nutritional reasons not related to neoplastic cellular changes, which is found in association with cirrhosis and malnutrition.

Sialadenitis: The gland can become enlarged from inflammatory conditions. These can be due to trauma to the maxillofacial area, viruses, or bacteria (*Staphylococcus aureus* most commonly), which can lead to abscess formation.

Unusual causes of enlargement have been found to be related to sarcoidosis, in which case multiple salivary glands may be found to be enlarged.

Sialolithiasis: Stones in the ducts of the salivary glands are a common condition leading to a mass within the gland or enlargement of the gland secondary to obstruction of salivary flow. Most of these are found in the submandibular gland and a lesser number in the parotid gland. A sialogram may sometimes show no stones but rather a stenosis from previous trauma that leads to obstructed or restricted flow.

Mucocele: These retention cysts of mucus production are most commonly found in the minor salivary glands. These are frequently located in the lower lip, and they sometimes develop after trauma to the lip, laceration and suture repair, or secondary healing.

Necrotizing sialometaplasia: This is an unusual and clinically disturbing lesion that has an uncertain cause. It may or may not be related to trauma to the oral mucosa but is manifest as an enlarging and often painless ulceration. It is more commonly found in the palatal mucosa but can also be seen in the buccal mucosa or the lips. It is a self-limited process and heals on its own, or it may be diagnosed by histologic examination after it is removed for not healing.

Benign neoplastic lesions:

Pleomorphic adenoma: Also known as the benign mixed tumour, this is the most common tumour of the salivary glands. The tumour was originally believed to be “mixed” neoplastic cells derived from both duct epithelial cells and myoepithelial cells. However, it is now known to be a neoplasm of purely ectodermal cells.

These tumours most commonly occur in the parotid glands (80 to 90%) and appear as painless,’ rubbery’ firm 1 to 2 cm masses in the deeper tissue. They are not attached to the overlying skin, and they do not cause any muscle dysfunction by pressure on the motor branches of the facial nerve. They can rarely be found bilaterally.

On histological examination, there is a distribution of mostly epithelial and stromal(mesenchymal) cells. These mesenchymal areas of the tumour may consist

of chondroid or hyalinized stroma that has the microscopic appearance of hyaline

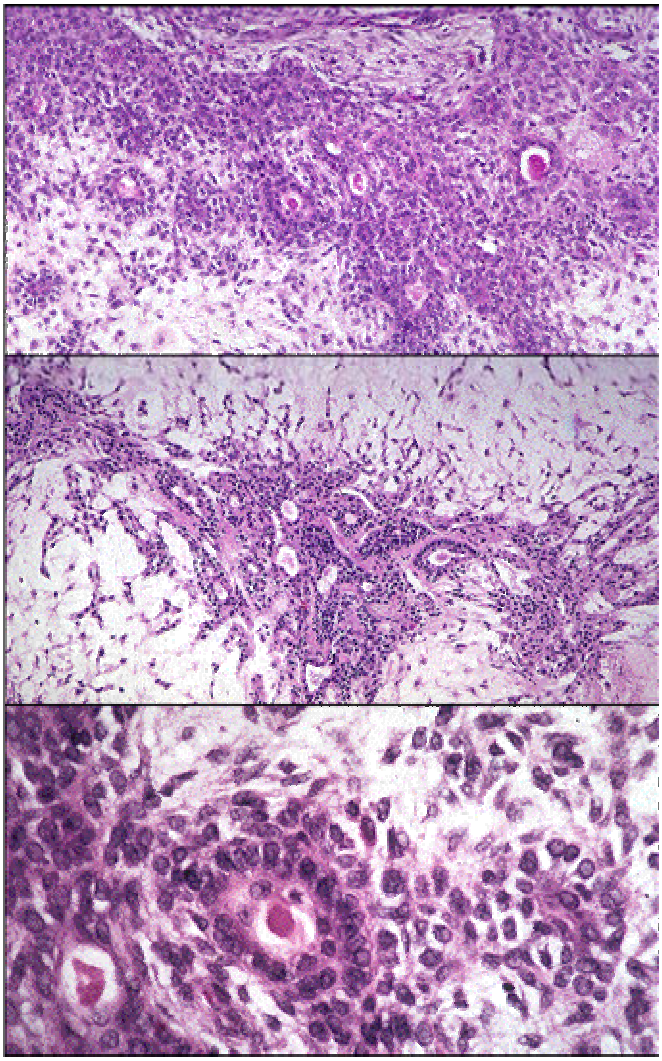


Figure 3. Mixed tumor

cartilage.

A preoperative diagnosis may be made by cytologic examination of a biopsy sample from FNA. The diagnosis is usually made after the tumour mass is removed with surrounding normal gland by means of resection of the entire segment of the parotid gland superficial to the plane of the facial nerve. The recurrence rate following this procedure is usually low in the range of 1%. On occasion, the

tumour is located in the deep segment of the parotid gland, requiring dissection of the facial nerve to protect the nerve and subsequently to remove the deep tumour.

Recurrent pleomorphic adenomas are uncommon but require careful management. They are best controlled by aggressive surgical removal and total parotidectomy when necessary to achieve long-term control rates of better than 90%. Postoperative radiation therapy has been shown to be effective in improving control rates in those patients at high risk for recurrence. Recurrence may also occasionally (less than 1%) be manifested as a transformation to malignant mixed tumour.

Warthin's tumour(papillary cystadenoma lymphomatosum): The second commonest benign tumour of the parotid gland, accounting for 6 to 10% of all parotid tumours. Found almost exclusively in the parotid, commoner in men in their fifth decade of life, it is clinically detected as a smooth mass, 3 to 4 cm in diameter, in the superficial segment of the gland and is bilateral in 10% of cases.

These tumours are derived from the proliferation of lymphoid tissues of periparotid or intraparotid lymph nodes. As such, they are believed to be proliferative tumours rather than neoplastic tumours. Grossly these tumours can have multiple cystic spaces and needle aspiration can yield a chocolate brown fluid. The tumour is well known for its "hot" appearance in technetium scans as it consistently concentrates this isotope. They are said to be associated with tobacco smoking and the increased incidence in females is attributed to the increasing use

of the same by females. Recurrence rates following surgical removal with a rim of normal tissue is extremely low and if present, signifies multicentric foci. Theories of origin of these tumours include 1. ectopic epithelial theory, 2. atypical hypersensitivity reaction theory, 3. branchial remnant theory.

Malignant neoplastic lesions:

"The usual tumour of salivary gland is a tumour in which the benign variant is less benign than the usual benign tumour and the malignant variant is les malignant than the usual malignant tumour" (Acherman L.V et al..)

Most malignant salivary gland tumours arise from either the excretory duct or the intercalated duct reserve cell. Either of these two cells have the potential for differentiation into a variety of epithelial tumours.

Mucoepidermoid carcinoma: It is the most common malignant tumour of the parotid gland and the second most common malignant tumour of the submandibular and minor salivary glands. The first reported case of mucoepidermoid carcinoma was in 1895, but it received its current name in 1945, based on the two main cellular components noted on microscopic examination- mucous cells and epidermoid cells. The palate is the second most common location for mucoepidermoid carcinomas. All mucoepidermoid tumours currently are believed to be carcinomas. Based on the relative ratios of the two cell types, they are divided into low, intermediate and high grades. Low-grade tumours have

numerous mucous cells and cystic spaces, whereas high-grade tumours may resemble a squamous cell carcinoma. 75% of these tumours are low grade.

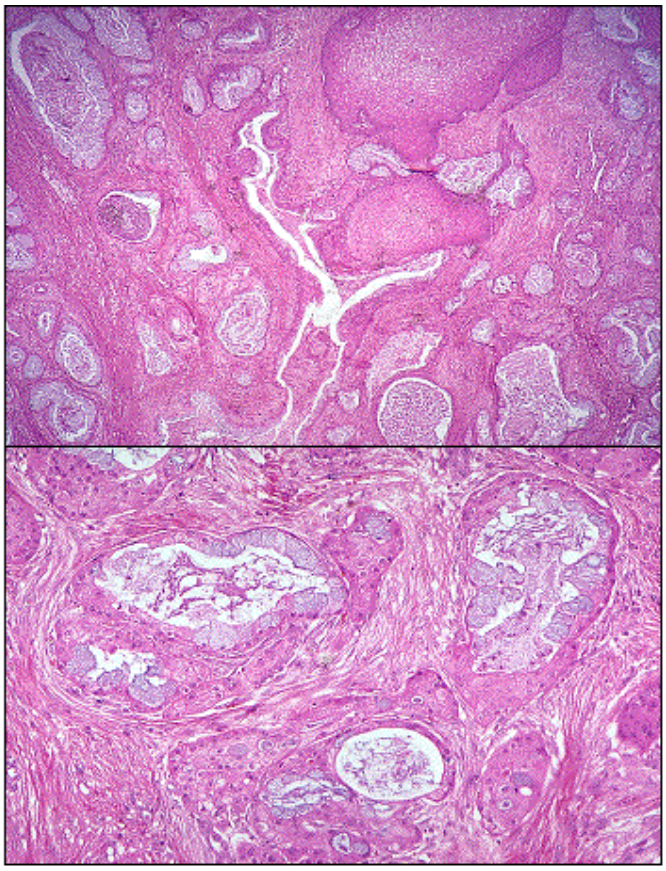


Figure 24. Mucoepidermoid Carcinoma

Microscopically the tumour is composed of varying percentages of six cell types. The maternal cell is the progenitor of the other five. The intermediate cell may differentiate into either glandular or epidermoid forms, and the epidermoid cell may resemble closely squamous cell carcinoma with individual cell keratinization, keratin pearls and intercellular bridges. The clear cell has a distinct outline with hydropic, water clear cytoplasm. The columnar cell resembles cells found in the major secreting ducts. The sixth cell is the mucous cell. Most low-

grade mucoepidermoid carcinomas present in a manner indistinguishable from pleomorphic adenoma.

Adenoid cystic carcinoma: The name adenoid cystic carcinoma was coined in 1953 by Forte and Frazell. This is the second most common malignant tumour of the salivary glands. The hallmark of this carcinoma is its propensity to perineural invasion.

There are three histological subtypes based on appearance: cribriform, tubular and solid. The cribriform pattern has the classic “Swiss cheese appearance” and in this type, cells are separated by round or oval spaces that appear to be cysts but are not. They are merely extracellular spaces lined by replicated basement membrane material. The tubular or “trabecular” pattern has a more glandular architecture, where as the solid pattern shows sheets of cells with little or no luminal spaces. The tubular variety has the best prognosis, where as the solid has the worst.

Malignant mixed tumour: Three different neoplasms have been included under the generic term malignant mixed tumour. Two are malignant from the beginning, the first of which is a histologically benign metastasizing pleomorphic adenoma. This lesion is a clinical and pathological curiosity. The second is a tumour with both a malignant epithelial component and a malignant myoepithelial component. In addition, the metastases contain both elements, and this lesion is a true carcinosarcoma. The third type, the carcinoma ex-pleomorphic adenoma,

represents the malignant transformation of the epithelial component within a pre existing pleomorphic adenoma. Only the epithelial component metastasizes. The risk of malignant transformation of a pleomorphic adenoma increases with the duration of the tumour and rises from about 1.5% within the first 5 years to 9.5% after 15 years. The sites of origin in decreasing frequency of carcinoma-ex-pleomorphic adenoma are the parotid, submandibular gland, palate, lip, paranasal sinuses, nasopharynx and tonsil.

Treatment

Benign tumours

The treatment of benign tumours of the parotid gland has passed through several phases during the past 30 years. Surgery is the treatment of choice for al salivary gland tumours.

Parotid gland: If it is a slow growing tumour, superficial conservative parotidectomy is the treatment of choice. If the tumour is present in one of the poles, then hemi superficial parotidectomy can be done. When the biopsy report turns out to be malignant then postoperative radiotherapy is advocated n a dose of 5000-6000 cGY in fraction over 5-6 weeks. Total parotidectomy is done for deep lobe tumours and dumbbell tumours.

Submandibular gland: Excision of the gland with preservation of lingual and hypoglossal nerve is advised.

Minor salivary gland tumours: After confirmation of clinical diagnosis by FNAC, excision of the tumour with wide margin of normal tissue is the treatment of choice. There is no need to sacrifice the underlying bone. The raw area may primarily be grafted with SSG or allowed to heal by granulation.

Intra operative issues:

From the onset, use of paralytics or muscle relaxants should be avoided by the anesthesiologist. This is done so that either electromyographic monitoring of the facial musculature or nerve localization by electrostimulation is permitted during the operation. The use of intraoperative facial nerve monitoring through the use of nerve integrity monitors can be done.

Hemostasis is improved with the use of bipolar cautery in areas adjacent to the facial nerve to help reduce thermal injury to the nerve. Recently, the Shaw scalpel has been used in parotidectomy to curb excessive blood loss and improve visualization in the operating field. the Shaw scalpel is a thermally activated scalpel, popularly known as the ‘hot knife’. Bipolar scissors with saline irrigation for faster dissection in a relatively bloodless field, harmonic scalpel all serve to improve intra-operative hemostasis

Complications of surgery:

Early:

1.Facial N neuropraxia	2.Facial N injury	3.Hemorrhage
4.Hematoma and seroma	5.Sialocele	6.Flap necrosis
7.Salivary fistula	8.Infection	9,Otitis externa

Late:

1.Frey ‘ s syndrome	2.Cosmetic deformity

Frey’s syndrome is the occurrence of redness and sweating on the temple in the area of distribution of the auriculotemporal nerve when eating, which is seen in few patients after parotidectomy. This is caused by regeneration of the secretory fibres of the parotid gland, contained in the auriculotemporal nerve, to sweat glands in its area of distribution. Thus the sweat glands respond to the nerve impulses that are meant to stimulate parotid secretion. It can be relieved by section of the IX nerve which carries the preganglionic fibres for the parotid gland(Gardner).

Facial nerve paralysis- treatment options:

- Neurorraphy
- Interposition nerve grafts
- Crossover reinnervation procedures

    i. Hypoglossal
    ii. Ansa hypoglossi
    iii. Cross facial
- Regional muscle transfer
    i. Temporalis
    ii. Massseter
    iii. Digastric
- Microneurovascular free flap
    i. Gracilis
    ii. Latissmus dorsi
    iii. Rectus abdominis
    iv. Serratus anterior
    v. Pectoralis major
    vi. Abductor hallucis
    vii. Extensor digitorum brevis

Static reanimation and cosmetic procedures:

- Eyelid procedures
    i. Gold weight
    ii. Spring
    iii. Lower lid tightening
- Brow & forehead lift
- Correction of midfacial deformity slings

i. Fascia lata

ii. Alloplastic sheets

iii. Malar augmentation

- Facelift operation
- Lower lip wedge resection
- Botulinum toxin

Adjuvant therapy:

Radiotherapy: The indications for and techniques of radiation therapy to be used as well as the response of salivary gland tumours to treatment depend upon the location of the primary lesions as well as extent and histologic make out. It is used as

Curative – as an adjuvant to surgery

Palliative – in inoperable tumours

for recurrent lesions

symptomatic treatment of distant metastasis

Indications for adjuvant radiotherapy to tumour bed are as follows:

- High grade histology(e.g.. mucoepidermoid carcinoma)
- All cases of adenoid cystic carcinoma
- Advanced stage of the primary
- Positive tumour margin

- Intraoperative tumour spillage
- Facial nerve involvement
- Positive neck nodes
- Deep lobe of parotid involvement

Since skip metastasis is possible, the entire neck should be irradiated. It is used as a dose of 6000-6500 cGy in fractionated doses over 6-7 weeks.

The complications of radiotherapy are:

Ageusia	Trismus	Diminished salivation
Caries tooth	Mucosal necrosis	Osteonecrosis

Chemotherapy: It is used as a palliative treatment for recurrent or metastatic salivary gland malignant neoplasms. Single drug therapy is commonly used with either one of the following- doxorubicin, 5-FU, methotrexate, cyclophosphamide, cisplatinum. Combination chemotherapy using regimens like CAP(cyclophosphamide, adriamycin, cis platinum) and FACP(5-FU,adriamycin,cyclophosphamide,cis-platinum)

Newer innovations: Advances in surgical techniques have enabled teams of head and neck surgeons and neurosurgeons to remove cancers that have spread near the base of the skull. These operations were not considered possible a few years ago but are becoming more common and successful.

Reconstructive surgery is becoming more sophisticated and successful. This permits more extensive surgery to be done and improves patient's' quality of life after treatment.

Advances in radiation therapy now permit more precise targeting of radiation. Some types of radiation, such as fast neutron beam radiation and conformal radiation therapy have been found to be particularly useful but require specialized equipment that is not available in many hospitals.

Researchers are learning more about how certain changes in DNA cause salivary gland cells to become malignant. For example, they have discovered that DNA from 2 chromosomes may undergo translocations (switch positions). These changes often cause activation of genes that control cell growth. For example, adenoid cystic carcinomas often have translocations involving chromosomes 6 and 9; in mucoepidemoid carcinomas, the translocations usually involve chromosomes 11 and 19. As scientists learn more about these and other DNA changes, they hope to use this information to develop new treatments for salivary gland cancers that are more effective and cause fewer side effects.

Because advanced salivary gland cancer is rare, knowledge about treating these cancers with chemotherapy is still evolving. New chemotherapy drugs and other anticancer drugs are expected to provide more options for people with advanced salivary gland cancer.

Targeted therapy: Drugs have been developed for other cancers that attack certain molecules in the cancer cell. This prevents the cancer from growing. These have

had some success in treating a host of other cancer types and are being tested in clinical trials in people with salivary gland cancer

Aims and objectives

The objectives of this study on surgical outcomes in salivary gland swellings are

(i)To assess the surgical outcomes of salivary gland surgeries

(ii)To evaluate the factors that contribute to the decision-making processes in salivary gland surgery

(iii)To document the various clinical presentations and the associated epidemiological values

(iv)To scrutinize and propose management protocols for the various surgically treatable disorders – benign, malignant and non-neoplastic – that affect these glands

(v)To document and deal with the complications which occur following surgery on these glands

Materials and methods

This study principally confined itself to those patients with salivary gland swellings, who were operated in Thanjavur medical college hospital between July 2005 and October 2007**.**

A meticulous proforma was prepared which included a thorough history, elaborate account of the clinical features and details regarding pre-operative investigations, operative notes and post-operative complications. All biopsy reports were collected and analysis done incorporating all these data. The final histopathological report was taken as confirmative of a salivary gland lesion and patients were excluded if the report did not confirm to be a salivary glandular lesion.

The pre-operative investigations included Fine Needle Aspiration Cytology of the lesion in all cases and CT scan, ultrasonography, sialogram were done for select cases. Other basic investigations for assessment purposes were done as per the institutional policies.

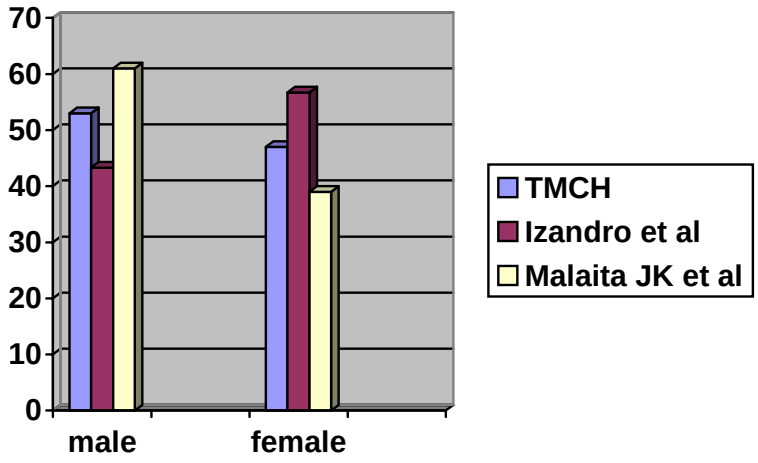
The study included only patients who were operated in our institution- under all the six general surgical units, plastic surgery, paediatric surgery and otorhinolaryngolgy units.

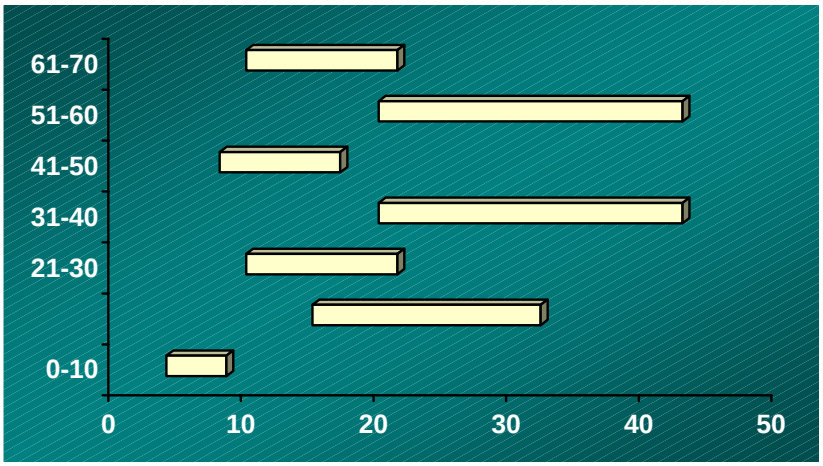
Observation and discussion

The total number of patients who had undergone surgery on salivary glands in the study period (July 05 to October 07) is ***87***. This group of patients was analyzed and the following inferences were obtained. This cohort of 87 proves a commendable number as the previous studies involving only neoplasms of the salivary glands in our institution give a rough incidence rate of 17 cases per year.

The distribution based on sex(values in percentages):

	TMCH	Izandro RB et al	Malaita JK et al
Male	53	43.3	61
Female	47	56.7	39
M:F ratio	1.12 : 1	1 : 1.3	1.5 : 1





There was no significant sexual predominance of salivary gland lesions which is also confirmed by certain other studies as shown in the table.

Age wise analysis:

The youngest among our patients was a 3 ½ year old child and the oldest was 70 year old females(2 patients).The distribution shows that majority of these lesions occur in middle-aged individuals. The mean age in our study was 39.6 years, the median age being 40 years.

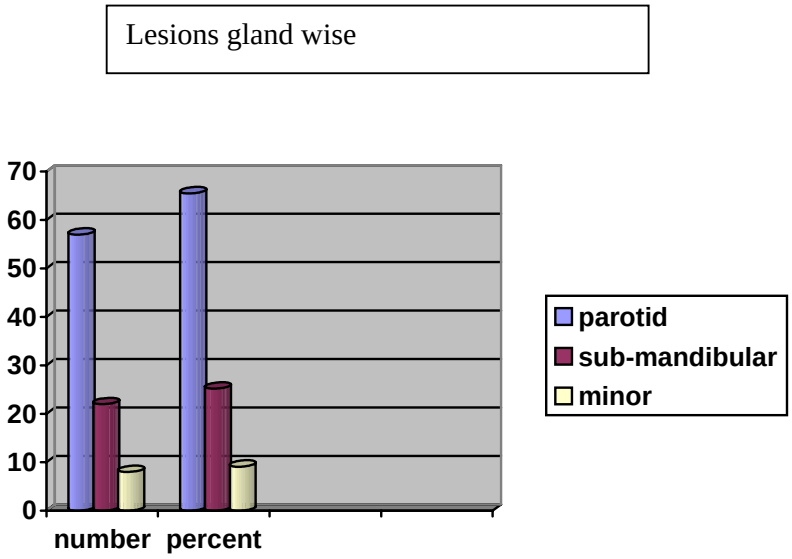
When distributed age wise,

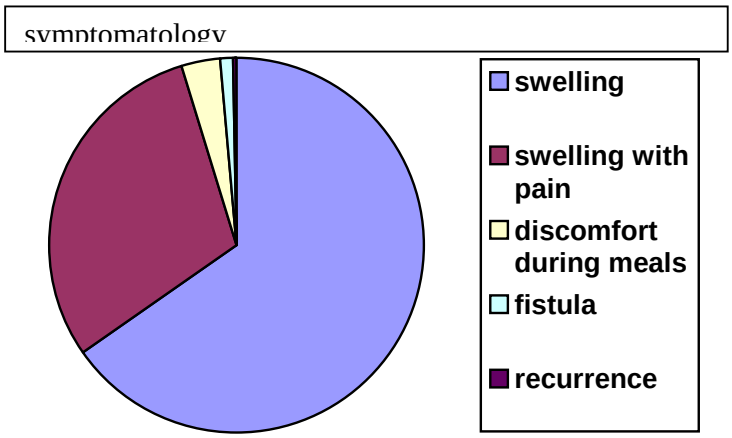
Age (in years)	Number	Percent
0-10	4	4.5
11-20	15	17.2
21-30	10	11.4
31-40	20	22.9
41-50	8	9.1
51-60	22	25.2
61-70	8	9.1

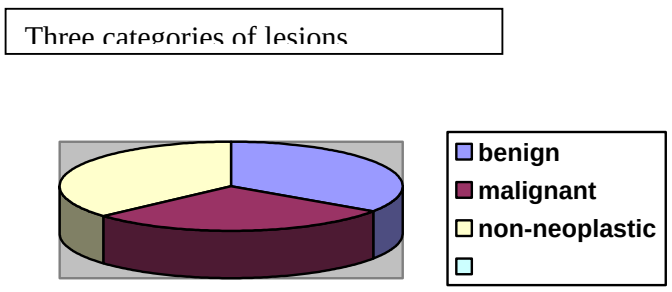
As would be expected the majority of the lesions were seen in the parotid glands – 57 cases in total and submandibular glands were affected in 22 patients and the rest 8 cases were of minor salivary gland lesions. When extrapolated into percentages this comes to be 65.51% parotid, 25.28% submandibular and 9.19% minor gland lesions. The sublingual and minor salivary glands are included in the same group. There was no separate distinctions among this minor gland group regarding their anatomical site. As is well known, the parotid gland lesions account for the maximum number of lesions followed by a considerable number of submandibular lesions. There were two cases of salivary gland tumours in cases clinically

diagnosed as carcinoma cheek as these lesions arose from glands located in the mucosal aspect of the cheek. The final histopathology report was considered the definitive inclusion criteria.

	Number	Percent
Parotid	57	65.51
Submandibular	22	25.28%
Minor	8	9.19%







No specific risk factors were identifiable and associations with smoking and tobacco use or past history of inflammatory disorders of these glands were not quantifiable or significant or both. There was no history of any previous irradiation in any of these patients.

Pathologically these lesions were grouped into three broad categories of benign, malignant and non-neoplastic, the distribution of which was:

Malignant 28.73%

Benign 34.48%

Non-neoplastic 36.78%

Individually the non-neoplastic lesions outnumber as the most frequently operated lesions but put together, benign and malignant neoplasms top the list. This sheds some light on the role of surgery in non-neoplastic lesions of the salivary glands.

Parotid:	57 cases		
	Benign	27	47.36%
	Malignant	16	28.07%
	Non-neoplastic	14	24.56%
Submandibular:	26 cases		
	Benign	5	23.8%
	Malignant	4	19%
	Non-neoplastic	12	57.1%
Minor/sublingual:	8 cases		
	Benign	1	12.5%
	Malignant	4	50%
	Non-neoplastic	3	37.5%

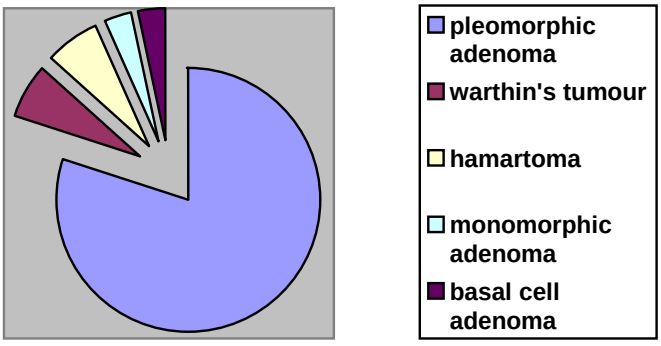
	Parotid	Submandibular	Minor
Benign	27	5	1

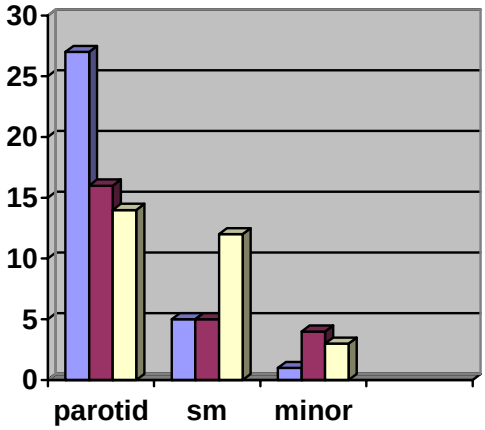
Malignant	16	5	4
Non-neoplastic	14	12	3

And the specific pathological types among benign, malignant and non-neoplastic lesions according to their frequency –

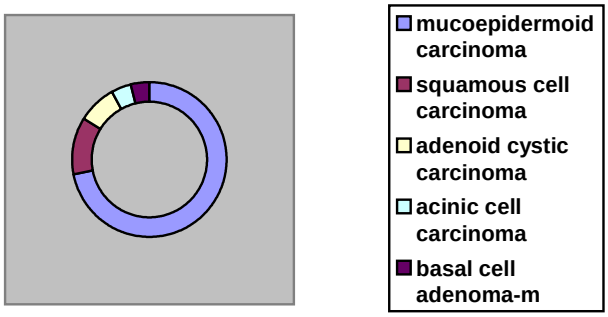
Benign: 30 cases

Pleomorphic adenoma	24
Warthin's tumour	2
Hamartoma	2
Monomorphic adenoma	1
Benign lesions	1





Gland-wise distribution



Malignant lesions

As can be seen pleomorphic adenoma accounts for about 80% of all benign lesions of salivary glands and most of them indisputably occur in the parotid gland.

Malignant: 25 cases

Mucoepidermoid carcinoma 18

Low grade 11

Intermediate 1

High grade 6

Squamous cell carcinoma	3
Adenoid cystic carcinoma	2
Acinic cell carcinoma	1
Basal cell adenoma-M	1

The predominance of mucoepidermoid carcinoma is no surprise though – but the occurrence of squamous cell carcinoma and basal cell adenoma with transformation into malignancy are slightly in discordance with various other studies in literature. The grading of mucoepidermoid carcinoma helps to ascertain adjuvant radiotherapy and also gives valuable prognostic information. One of these patients in our study was pre operatively diagnosed as pleomorphic adenoma while the HPE report was that of mucoepidermoid carcinoma(high grade) and was then registered for radiotherapy, and the same patient developed sialocele as a post operative complication.

The non-neoplastic swellings were 32 in number which on HPE :

Non specific inflammation	17
Chronic sialadenitis	12
Granulomatous	2
Cyst	1

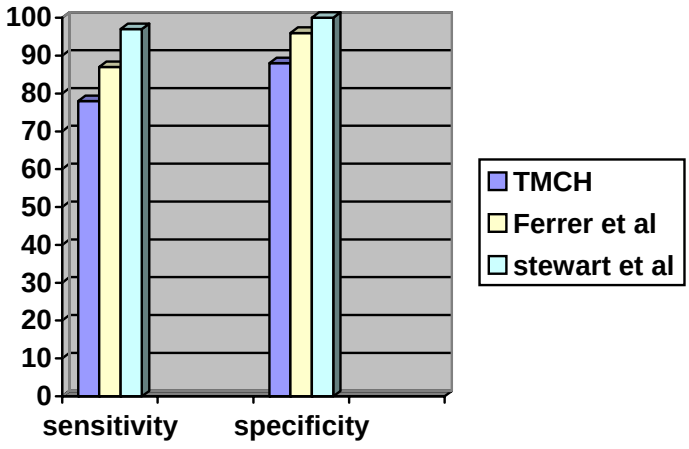
Most of these non-specific inflammations showed congestion and other features of inflammation with varying amounts of destruction of glandular architecture and were found unrelated to calculous diseases. The second in the list, of chronic sialadenitis included sialoliths also and these lesions were found predominantly in submandibular salivary gland. Of the two cases of granulomatous lesions one was suggestive of tuberculosis, and the patient was registered for anti-tuberculous therapy, but unfortunately could not be followed up. And the cystic lesion was a simple cyst with salivary glandular epithelial lining.

The intraoperative issues were usually the careful identification and preservation of facial nerve and its branches. There was one case in which the main trunk of the facial nerve had to be sacrificed due to tumour infiltration though the patient did not have any clinically demonstrable nerve deficits preoperatively. There were no radical parotidectomies done in the study period and those patients with facial nerve involvement were not operated in our institution during the study period – often due to the unwillingness of these patients when explained that the recovery chances of the neurological deficit were not high. And those patients who had post-operative facial nerve weakness, no obvious clues were identified intra-operatively. There were 7 such patients in our study out of which 4 improved with conservative treatment, and the other three had to be taught the necessary protective measures.5 of these 7 patients were cases of malignant lesions and 1 benign and 1

inflammatory lesion. And the recovered 4 cases were again 2 malignant, 1 benign and 1 inflammatory lesion. No reparative surgery was undertaken for any of these patients during the study period.

	Benign	Malignant	Non-neoplastic
Facial N palsy	1	5	1
Resolved spon.	1	2	1

The role of FNAC was also analyzed and it was found that about 20 cases (22.98%) proved to be different from the final tissue diagnosis. The false negative rate was higher than false-positive rate in case of neoplasms. The commonest report on FNAC was pleomorphic adenoma and 10 of these cases turned out in the end to be different lesions. The sensitivity and specificity of this modality of investigation was computed and is as shown in the graphic representation.

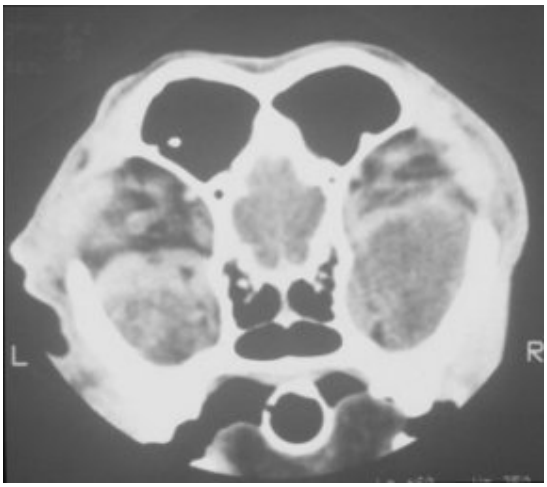


Of the 87 cases of FNAC reports, about 20 cases proved different from the final histopathological report. There were 4 false positive cases, 11 false negative cases. True positives were about 40 and true negatives 32 in our study. Taking these values and computing sensitivity and specificity, our study shows FNAC to be 78% sensitive and 88% specific.

Sialogram showing left parotitis

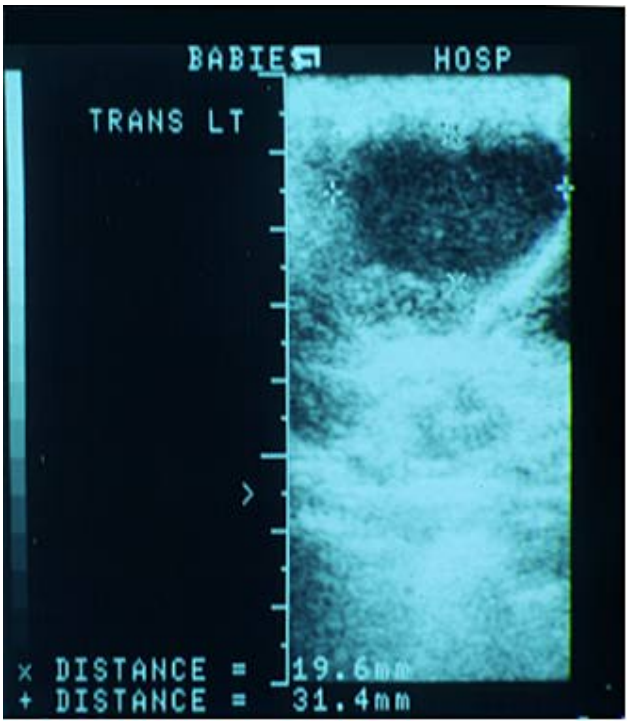
Investigations like sialogram, ultrasonogram and CT scan were done in a few select cases as and when appropriate. Though these investigations proved to be more

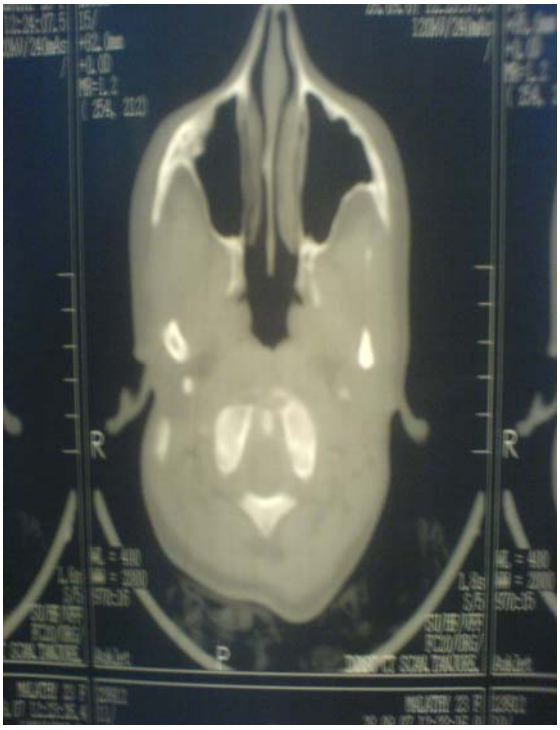
informative they cannot be indicated in all cases considering the cost-benefit ratio, hence these modalities of investigations were not routinely done in all patients.



CT scan showing Left Parotid swelling

Ultrasonogram of salivary gland swelling



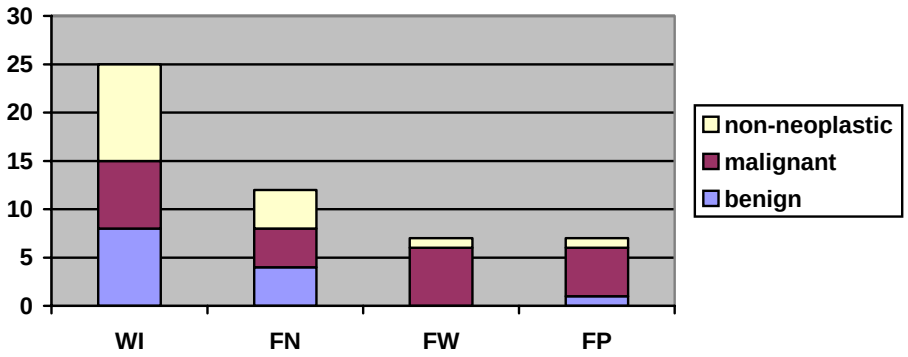


CT scan parotid

Complications which occurred in the post-operative period were documented and analyzed. The list included facial nerve weakness, facial nerve palsy, flap necrosis,

wound infection, salivary fistula, sialocele and recurrence. Dysgeusia and dryness of mouth was reported by some patients but these symptoms spontaneously

resolved and were not serious enough to be



considered.

The complications met with other than facial palsy were that of flap necrosis which occurred in 12 of our patients- 4 each in the three pathological categories- accounting for about 13.9%. The next in the list was wound infection which was seen in 25 patients ranging from seropurulent discharge to wound gaping, this was seen more with non-neoplastic lesions than neoplastic lesions-in total of about 20.8% of all the cases. Facial nerve weakness which resolved spontaneously and/or with steroids was observed in 7 cases – 6 of them being malignant lesions. This was numerically only 8.04%, and the average duration for this recovery was about 16days. Rare complications like sialocele was seen in one patient, recurrence noticed in few cases but the primary surgery in these patients had been done earlier than the study period. Frey's syndrome was not encountered with in any of these patients.

All of these patients were discharged within two weeks when the minor complications if any were taken care of. They were asked to attend the review opd the following week when the biopsy report was ready and subsequent categorizations done accordingly. Those patients who required adjuvant therapy were then referred to radiotherapy department and followed up from there. Those who needed supportive treatment till complete remission of their otherwise innocuous symptoms were asked to attend the review opd weekly from then. Most of these patients were regular in their follow up and were able to give valuable information on their improvement or newer complaints, if any.

However, this study and the analysis has its own limitations in that no radical surgery was done in the study cohort, no cases of facial nerve palsy were operated upon and neither was any neck dissection done. No cases of lesions of deep lobe of parotid were operated upon. Also the patients did not adhere to the follow-up protocol which made it difficult in few cases to document the results.

Conclusions

The analysis of our study cohort leaves us with the following inferences:

- There is a great deal of surgical work to be done and is being satisfactorily done on salivary gland swellings in our institution.
- The overall outcome remains considerably good both for the patient and the operative surgeon.
- The role of surgery in non-neoplastic swellings is well made out and brought to light in this study.
- The gland affected more is the parotid gland and the most common lesion is pleomorphic adenoma.
- There is no sexual predominance in these lesions and the age incidence is higher in middle aged adults.
- FNAC still proves to be complementary and the choicest modality with a sensitivity of 78% and specificity of 88%
- The rate of complications are within acceptable limits and the patients are not disabled in the long-term analysis
- The knowledge on radical surgeries and facial nerve repair is lacking in this study period and needs to be performed more in the near future
- The numerical results of the various parameters analyzed prove to be in concurrence with medical literature and other studies

In short, this study brings it to the forefront that salivary gland surgery-a demanding and craftful field considered as rarely performed- is in truth not so rare. The number and variety of cases in this particular surgical pathology is considerable and the results are laudable, the only hitch being the reluctance to perform radical surgery especially due to the limitations of investigating modalities. This study is not a path-breaking one but is a useful signpost for the future advancements in salivary gland surgery in our institution.

clinical pictures of salivary gland swellings

operative and post operative pictures

MASTER CHART

S.NO	NAME	AGE/SEX	I.P.NO..	SYMP	GLAND	VIIN	FNAC	SURGERY	HPE	COMP	ADJ
1	SANTHANAM	57/M PS	838160	S	M	-	I	E	I	-	-
2	VEERAMMAL	35/F S2	839131	S	P	-	P	SP	P	WI	-
3	KALIYAPERUMAL	57/M S4	837272	S/P	P	-	MEC	TCP	MEC-L	FN	+
4	AYYASAMY	47/M S2	840223	S	P	-	P	SP	P	-	-
5	CHELLATHURAI	50/M S6	840363	S/P	P	+	MEC	TCP	MEC-H	FW	+
6	AMSAVALLI	35/F S5	839728	S/P,D	S	-	CSA	E	CSA	NW	-
7	KOTTU	60/F S3	840950	S	P	-	W	SP	W	-	-
8	RAJESWARI	40/F S1	842992	S	P	-	P	SP	P	FN	-
9	SARATHA	60/F S1	844696	S	P	-	P	SP	P	-	-
10	*MALAIAMMAL*	*60/F S6*	*846763*	*S*	*S*	-	*P*	*E*	*P*	*WI*	-
11	AMSU	15/F S2	846686	O	P	-	CSA	SP	CSA	WI	-
12	SAMIAPPAN	42/M PS	868191	S/P,F	P	-	I	SP	I	FN	-
13	VERAN	30/M PS	869214	S/P	P	-	CSA	SP	CSA	FW	-
14	RAMAYEE	40/F S3	870556	S/P	P	-	BCA	SP	BCA	WI	-
15	PATTAMMAL	70/F S5	870015	S,O	P	-	MEC	TCP	MEC-H	WI	+
16	ALLAPITCHAI	60/M S1	872054	S/P	S	-	CSA	E	SCC	WI	+
17	SIVARAMAN	42/M S6	874356	S	P	-	P	SP	P	FN	-
18	MARIMUTHU	70/F S2	875561	S/P	P	-	M	SP	M	-	-
19	LOGAMBAL	70/F S4	876011	S	P	-	P	SP	P	WI,FP	-
20	KITTURAR	57/M S4	876681	S	P	-	I	SP	I	WI	-
21	BHUVANESHWARI	24/F S6	876064	S	P	-	P	SP	P	-	-
22	CHANDRASEKARAN	23/M S1	876790	S	P	-	P	SP	MEC-L	FN	+
23	VADIVEL	60/M S4	878477	S/P	S	-	CSA	E	CSA	WI	-
24	VAHITHA BEGUM	32/F S2	878244	S	P	-	P	SP	MEC-L	FW	-
25	THANGAVEL	60/M S4	878978	S/P	P	-	MEC	TCP	MEC-H	WI	+
26	JEYAKUMARI	33/F S2	879989	S/P	P	-	P	SP	I	WI	-
27	PARTHIBAN	35/M S3	879693	S,LN	P	-	P	SP	P	WI	-
28	DHANASEKAR	38/M PS	879844	S	M	-	SCC	WE	SCC	-	+
29	KALIYAMOORTHY	19/M S3	880063	S/P	S	-	I	E	G-TB	WI	+
30	RAMKUMARI	62/F S1	880592	S	P	-	P	SP	P	-	-
31	AARTHY	18/F ENT	11286	S	P	-	P	SP	P	WI	-
32	NIRMALA	30/F S2	883329	S/P	S	-	CSA	E	CSA	WI	-
33	SAROJINI	23/F S6	884329	S	P	-	P	SP	ACC	-	-
34	KALAIVANAN	17/M S5	885071	S,O	S	-	CSA	E	CSL	-	-
35	MANIKANDAN	15/M S1	885844	S/P	P	-	MEC	TCP	MEC-L	WI,FP	+
36	GUNASUNDARI	40/F ENT	110841	S,R	P	-	P	TCP	P	-	-
37	RAJENDRAN	42/M S1	886318	S/P	P	-	I	SP	C	FN	-
38	SATHYA	12/F P	180667	S	M	-	I	E	MEC-L	FW	+
39	BHAKYAVATHY	8/F P	183652	S/P	S	-	CSA	E	G	WI	-
40	SELVA ANAND	28/M S5	891600	S	P	-	P	SP	P	-	-
41	VELMURUGAN	35/M S6	901280	S/P	P	-	P	SP	MEC-L	FW	+
42	SARATHKUMAR	3 ½ /M P	176294	DI	P	-	I	SP	G	WI	-
43	THANGARASU	60/M S2	901361	S/P	P	-	P	SP	P	-	-
44	SAVARIRAJAN	33/M S4	901993	S	M	-	I	E	I	-	-
45	NALLAMMAL	60/F S5	902866	S/P	P	-	I	SP	I	-	-
46	MUTHUKUMAR	55/M S1	939104	S,R	P	-	MEC	TCP	MEC-I	WI,FP	+
47	SUNDARAMBAL	40/F S1	903096	S,R	P	-	MEC	TCP	MEC-L	FW	+
48	MURUGARAJAN	62/M S4	904591	S	S	-	I	E	I	-	-
49	DURAIRAJ	31/M S5	906103	S/P	P	-	P	SP	I	-	-
50	SHANKAR	5/M P	118874	DI	P	-	I	SP	I	-	-
51	DHIVYA	12/F S4	9067568	S	S	-	P	E	P	-	-

52	LAKSHMANAN	28/M S3	926062	S/P	P	-	MEC	TCP	MEC-L	-	-
53	RAMAIYAN	44/M S2	927011	S/P,O	P	-	I	SP	I	WI	-
54	CHANDRA	40/F S2	928193	O	M	-	ADC	E	ADC	FW	-
55	KALIYAPERUMAL	55/M S3	927301	S/P,O	S	-	CSA	E	CSA	FP	-
56	ELANCHIYAM	20/M S6	928704	S	P	-	P	SP	P	-	-
57	RANJITHAM	29/F S3	929464	S	P	-	P	SP	W	WI	-
58	MALAIYAMMAL	66/F S3	929804	S	S	-	P	E	P	-	-
59	RAMACHANDRAN	34/M S3	931223	S	P	-	P	SP	P	-	-
60	JENNIFER	19/F S2	932323	S,LN	S	-	MEC	E	MEC-L	-	+
61	KANNU	60/F ENT	07988	O	M	-	I	E	ADC	-	-
62	SAMBANDHAM	57/M S3	934579	S	P	-	P	SP	P	-	-
63	PUSHPAVALLI	37/F S1	933883	S/P	P	-	P	SP	I	FN	-
64	SARAVANAN	18/M S4	932999	S,LN	S	-	I	E	I,H	-	-
65	ANJALAI	60/F S5	941574	S	P	-	P	SP	P	-	-
66	SARAVANAN	36/M S3	943199	S	P	-	P	SP	P	-	-
67	AROKIASAMY	35/M S5	942605	S/P	S	-	CSA	E	CSA	WI	-
68	SHANKAR	36/M S1	944224	S/P	P	-	MEC	TCP	BCA-M	FN	-
69	RAMAIYAN	54/M S5	947929	S/P,LN	S	-	MEC	E	SCC	WI,FP	+
70	SATHYA	20/F ENT	32589	S/P	S	-	CSA	E	CSA	FN	-
71	DEVASAGAYAM	65/M S1	954118	S/P	P	-	P	SP	MEC-H	-	+
72	RAMANATHAN	20/M ENT	39638	S	P	-	P	SP	P	-	-
73	SARAVANAN	20/M S4	938475	S/P	P	-	I	SP	I	-	-
74	CHANDRA	65/F ENT	0309607	S	P	-	P	SP	I	WI	-
75	JEYALAKSHMI	48/F S4	938977	S	P	-	I	SP	C	-	-
76	MUTHUKUMAR	55/M PS	908311	S/P	P	-	P	SP	MEC-I	FN	+
77	MARIYAMMAL	40/F S3	913018	S/P,O	S	-	I	E	I	-	-
78	MANICKAM	55/M S5	917304	S/P	S	-	MEC	E	MEC-L	-	+
79	MARUTHAMBAL	63/F S5	917312	S/P	P	-	I	SP	P	-	-
80	MALAIYAPPAN	63/M S2	919581	S/P,LN	S	-	CSA	E	CSA	-	-
81	ANITHA	4/F P	48284	S	P	-	H	SP	H	-	-
82	ANJALAI	33/F S4	920404	S	P	-	P	SP	P	-	-
83	BALAMURUGAN	41/F S3	920673	DI,O	S	-	I	E	I	-	-
84	SAKTHIVEL	12/M P	49444	S	P	-	P	SP	MEC-L	FP	+
85	SABAPATHY	60/M S3	927341	S	S	-	MEC	WE	SCC	WI	+
86	RANI	20/F S3	928436	S	P	-	P	SP	P	FN	-
87	MALATHY	27/F S6	928464	S,R	M	-	P	E	P	FN	-

ABBREVIATIONS:

SYMPTOMS:

S	-	SWELLING
S/P	-	SWELLING WITH ASSOCIATED PAIN
O	-	OTHERS
DI	-	DISCHARGE

GLAND:

P	-	PAROTID
S	-	SUB MANDIBULAR
SL	-	SUB LINGUAL
M	-	MINOR SALIVARY GLANDS

FNAC / HPE:

P	-	PLEOMORPHIC ADENOMA
MEC	-	MUCO EPIDERMOID CARCINOMA(L- LOW, I- INTERMEDIATE, H- HIGH)
M	-	MONOMORPHIC ADENOMA
W	-	WARTHIN'S TUMOUR
ACC	-	ACINIC CELL CARCINOMA
ADC	-	ADENOID CYSTIC CARCINOMA
SCC	-	SQUAMOUS CELL CARCINOMA
BCA	-	BASAL CELL ADENOMA
BCA-M	-	BASAL CELL ADENOMA WITH MALIGNANT TRANSFORMATION
H	-	HAMARTOMA
CSA	-	CHRONIC SIALADENITIS
G	-	GRANULOMATOUS
C	-	CYST
I	-	INFLAMMATORY

SURGERY:

E	-	EXCISION
WE	-	WIDE EXCISION
SP	-	SUPERFICIAL PAROTIDECTOMY
TCP	-	TOTAL CONSERVATIVE PAROTIDECTOMY

COMPLICATIONS:

FN	-	FLAP NECROSIS
WI	-	WOUND INFECTION
FW	-	FACIAL NERVE WEAKNESS
FP	-	FACIAL NERVE PALSY

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PROFORMA

NAME: ***AGE:*** ***SEX:***
ADDRESS: ***IP NO..:***

OCCUPATION:

COMPLAINTS

SWELLING:

SITE:
DURATION:
RATE OF GROWTH:
ANY DISCHARGE:
IF YES, NATURE:
SUDEN CHANGE IN SIZE:

PAIN:

OVER THE SWELLING:
REFERRED PAIN TO EAR:
PAIN DURING MASTICATION:

ANY INCREASED SALIVATION
LOSS OF APPETITE/WEIGHT
NERVE INVOLVEMENT

PUS DISCHARGE-FROM SITES OF DUCTAL ORIFICES
BLOOD STAINED SALIVA
H/O SMOKING
ALCOHOL
TOBACCO CHEWING
H/O IRRADIATION
ANY FAMILY HISTORY

CLINICAL EXAMINATION

SWELLING

SITE *LIFTING OF EARLOBULE* *SIZE*
SURFACE *SHAPE* *DISCHARGE*
MOBILITY *SKINCHANGES* *WARMTH*
TENDERNESS
PLANE OF THE SWELLING
BIDIGITAL PALPATION
SWELLING IN THE FLOOR OF MOUTH, ORAL CAVITY
NERVE INVOLVEMENT
CERVICAL LYMPHADENOPATHY

INVESTIGATIONS

FNAC
CT SCAN
USG
SIALOGRAM
OTHERS FOR ASSESSMENT PURPOSE

TREATMENT

SURGERY - *SP,TCP,E,WE*
RT
ATT

POST OPERATIVE PERIOD

FLAP NECROSIS
WOUND INFECTION
FACIAL NERVE WEAKNESS
FACIAL NERVE PALSY
SIALOCELE
SALIVARY FISTULA

RECURRENCE